Noninsertional Achilles Tendinopathy Pathologic Background and Clinical Examination

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KEYWORDS

• Achilles • Tendinopathy • Tendinosis

KEY POINTS

- Achilles tendinopathy includes a spectrum of histologic pathologic findings ranging from acute inflammation to chronic degeneration and fiber rupture.
- Tendinopathy is a clinically diagnosed condition with specific clinical cues leading to accurate diagnosis.

INTRODUCTION

The term tendinopathy includes a series of pathologies, all of which have a combination of pain, swelling, and impaired performance.[1] Most authorities advocate the use of the term tendinopathy to encompass each of the subclasses of Achilles tendon pathology. The terms tendinosis, tendinitis, and peritendinitis are all within the main heading of tendinopathy, and this terminology provides a more accurate understanding of the condition and highlights the uniformity of clinical findings while distinguishing the individual histopathological findings of each condition.[2,3] Understanding both the clinical features and the underlying histopathology leads to a more accurate clinical diagnosis and subsequent treatment selection.

This is an important distinction, because the misuse of the term tendinitis in the clinical diagnosis and treatment of these disorders can lead to the underestimation of chronic degenerative nature of many tendinopathies, which may affect the treatment selection.[2] Specifically, treating the chronic degenerative forms of tendinosis with immobilization and anti-inflammatory medications commonly used for acute inflammatory processes may lead to treatment failures and have the potential to drive

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unnecessary surgery.[4] The treatment of these pathologies, namely noninsertional tendinosis, will be discussed in subsequent articles and is guided by an understanding of the underlying pathologic process.

The basic etiology of the Achilles tendinopathy is known to be multifactorial. The pathophysiology of chronic Achilles tendinopathy is thought to involve the cellular and molecular response to microscopic tearing of the tendon when forces beyond the elastic capabilities of the tendon are applied to the tissues leading to chronic degeneration. Histologic examination of the affected tissue demonstrates an irregular shape and a higher rate of apoptosis.[5–7] Although inflammation occurs around the tendon, biopsies demonstrate no inflammatory cells infiltrating the tendon. Tendinopathy is understood as a failed healing response within the extracellular matrix that is mediated by a cascade of proinflammatory molecules that include interleukin-1B, prostaglandin E2, and nitric oxide.[8,9] In patients who develop tendinopathy, these mediators induce apoptosis, signal pain responses, and increase the production of matrix metalloproteinases (MMPs).[7] This response leads to degeneration of the tendon, rather than signaling a repair process.[8,10]

Histologic evaluation of tissue taken from ruptured Achilles tendons has been shown to contain more degeneration than those taken from patients with tendinopathy and uninvolved controls.[11,12] A similar study revealed that almost all of the Achilles tendons operated on for rupture showed signs of hypoxic degenerative tendinopathy, calcifying tendinopathy, mucoid degeneration, or tendolipamatosis.[13] In a large retrospective case-control study by Tallon and colleagues,[14] no spontaneous Achilles tendon ruptures were found in patients with healthy tendons. It is important to note, however, that Achilles tendon ruptures can take place suddenly without any preceding signs or symptoms.[12]

Because the Achilles tendon is the strongest and thickest tendon in the body and is subjected to unique forces during the activities of living, it is highly subject to tendinopathy, which can ultimately result in chronic degenerative changes, as well as calcification and mucoidlike degeneration, leading to Achilles tendinosis.[12] Because of the unique anatomy of the Achilles tendon, including its rotational change with spiraling proximal to its insertion, the Achilles is under significant biomechanical strain 2 to 6 cm proximal to its insertion into the calcaneus. While the degeneration at this area has been attributed to avascularity, this may not be the case, as discussed in this issue (see Paul Dayton's article, "Anatomic, Vascular, and Mechanical Overview of the Achilles Tendon," in this issue).

As noted previously, several theories exist regarding the etiology of Achilles tendinopathy. These include overuse, poor tissue vascularity, mechanical imbalances of the extremity, and a genetic predisposition.[4,15,16] Tendinopathy secondary to overuse is thought to arise from repetitive microtrauma in the central portion of the tendon. A retrospective case-control study identified several patient factors that were more likely to be associated with Achilles tendinopathy: hypertension, diabetes, obesity, and a previous exposure to steroids or estrogen. Each of these factors has the potential to decrease the microvascularity of tendons and as such have been postulated to play a role in the development of Achilles tendinopathy.[17] Other studies have found advancing age, previous injury, exposure to quinolone antibiotics, and endocrine and metabolic abnormalities to be associated with Achilles tendinopathy.[18–20] From a biomechanical standpoint, Williams and colleagues[21] found patients with Achilles tendinopathy to have decreased tibial external rotation during running, which was attributed to an imbalance of muscle forces in the transverse-plane of motion that increases the strain on the Achilles tendon. Finally, the gene for matrix metalloprotease-3 (MMP-3) is involved in the homeostasis of the ground substance surrounding